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EXAMINER	
UNGAR, SUSAN NMN	

ART UNIT	PAPER NUMBER
1642	

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Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/018,396	CHO-CHUNG, YOON S.	
	Examiner	Art Unit	
	Susan Ungar	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *hree* MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on *10 January 2005*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☐ Claim(s) *1-20* is/are pending in the application.

4a) Of the above claim(s) *10-20* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☐ Claim(s) *1-9* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *2/8/02*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

1. The Election filed January 10, 2005 in response to the Office Action of December 9, 2004 is acknowledged and has been entered. Claims 1-20 are pending in the application and Claims 10-20 have been withdrawn from further consideration by the examiner under 37 CFR 1.142(b) as being drawn to non-elected inventions. Claims 1-9 are currently under prosecution.

2. Applicant's election with traverse of Group 1, claims 1-3, 4-in-part, 5-8 is acknowledged. The traversal is on the ground(s) that under PCT Rule 13.2, a group of inventions is considered linked to form a general inventive concept where there is a technical relationship among the inventions that involves at least one common or corresponding special technical feature. Further, PCT Rule 13.2 defines the term "special technical features" as meaning technical features that define a contribution, which each claimed invention, considered as a whole makes over the prior art. Applicant argues in particular that the restriction requirement is improper because the claims are so linked as to form a single general inventive concept since all of the pending claims involve ECPKA protein and cancer. The argument has been considered but has not been found persuasive because as previously set forth, the claimed inventions do not form a general inventive concept because the claims are all drawn to a single category, that is method claims, and the claims do not have unity of invention because they are not drawn to any of the five category groups recited previously. It is suggested that Applicant review PCT article 17(3) (a) and 1.476 (c), 37 C.F.R. 1.475(b) and (d) for further information.

Applicant further argues that a search for prior art with respect to any of the groups would likely uncover references that would be considered by the Examiner during the examination of the other groups and Applicant describes similarities

between the groups. Applicant appears to be arguing that search of all of the groups would not be an undue burden on the Examiner. As drawn to claims 4 and 9, the argument has been considered and has been found persuasive and claims 4 and 9 have been rejoined with the Group 1. However, as drawn to the other claims and groups, the argument has been considered but has not been found persuasive because the literature search, particularly relevant in this art, is not coextensive and different searches and issues are involved in the examination of each group. For these reasons the restriction requirement is deemed to be proper and is therefore made FINAL.

Claim Rejections

Claim Rejections - 35 USC § 112

3. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

4. Claims 1-9 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of diagnosing carcinoma in a patient comprising assaying a fluid sample from the patient for increased phosphorylation activity, does not reasonably provide enablement for a method of diagnosing cancer in a patient comprising assaying a sample from said patient for elevated level of ECPKA/assaying with antibody to catalytic subunit of ECPKA/assaying with antibody to the regulatory subunit of ECPKA, wherein the cancer is breast cancer, prostate cancer, ovarian cancer, colon cancer, pancreatic cancer, lung cancer or bladder cancer. The specification does not enable any

person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims.

The claims are drawn to a method of diagnosing cancer in a patient comprising assaying a sample from said patient for the elevated presence of ECPKA/assaying with antibody to catalytic subunit of ECPKA/assaying with antibody to the regulatory subunit of ECPKA wherein the presence of an elevated level of ECPKA in said sample compared to the level of ECPKA in a control sample is indicative of cancer in said patient. This means diagnosing any cancer by detecting overexpression of ECPKA protein in any sample, this means assaying specifically for cancer is breast cancer, prostate cancer, ovarian cancer, colon cancer, pancreatic cancer, lung cancer or bladder cancer.

The specification teaches that in mammalian cells, there are two types of PKA, type I (PKA-I) and type II (PKA-II) which share a common C subunit but contain distinct R subunits, RI and RII respectively. Through biochemical studies and gene cloning, four isoforms of the R subunits, RIalpha, RIbeta, RIIalpha and RIIbeta have been identified. Further three distinct C subunits, Calpha, Cbeta and C gamma have also been identified (p. 2, lines 24-31) wherein preferential co-expression of one of these C subunits with any of the R subunits has not been found. The specification further teaches that it has been surprisingly and unexpectedly discovered than an ECPKA exists and that its presence reflects cell transformation resulting from the loss of regulation of growth (p. 3, lines 25-30), that is, that it is a novel PKA. The specification further teaches that a monoclonal antibody that distinguishes ECPKA from intracellular PKA and ectoPKA can be generated in accordance with methods known in the art (p. 20, lines 25-28). Further, the specification demonstrates that ECPKA is immunologically related to

intracellular PKA (see pages 25-29 and page 33). The specification teaches that ECPKA phosphorylates Kemptide, a standard assay substrate for PKA (p.22, lines 1-15) and is inhibited by PKI, a standard PKA phosphorylation inhibitor. ECPKA is a Type I PKA based on 8-Cl-cAMP assay (p. 25, lines 25-27) and based on Western Blot comparisons with known PKA subunits (p. 33, lines 9-26).
The specification teaches that the present invention provides a method of diagnosing cancer in a patient comprising assaying for the presence of ECPKA. The specification further teaches that the specific type of cancer detected by the presence of ECPKA can be subsequently or simultaneously determined by methods well-known in the art and teaches well known genetic or protein markers which are predictive of a particular cancer (p. 10). It is noted however that the claims as currently constituted are not limited to assays including the subsequent or simultaneous determination of specific cancer type.

Further, while any sample from the patient theoretically can be used in the assay, desirably the sample is a fluid sample (p. 9, lines 1-15).). Serum samples of cancer patients were assayed for ECPKA phosphorylation activity with the Kemptide assay wherein it is exemplified that ECPKA activity was significantly elevated in the serum samples from carcinoma cancer patients compared to that in normal serum samples (p. 34), wherein the ECPKA activity was not stimulated with cAMP but was inhibited by the PKA inhibitor PK1 which indicates that the ECPKA in human sera was present in the active free C subunit form (p. 35).

(A) As drawn to assaying ECPKA protein concentration, one cannot extrapolate the teaching of the specification to the scope of the claims because although the specification describes the similarities of ECPKA to intracellular PKA and teaches that monoclonal antibody that distinguishes ECPKA from

intracellular PKA and ectoPKA can be generated in accordance with methods known in the art, no distinguishing structure for ECPKA is taught by the specification that would enable one of ordinary skill in the art to produce an antibody or any other molecule that could predictably distinguish between ECPKA and other forms of PKA in order to predictably diagnose cancer by assaying for ECPKA protein in, for example, a solid tissue sample given that the activity of the "novel ECPKA" appears to be the same or similar to known PKAs. It appears that Applicant is claiming the assay of a novel functional equivalent of known PKA by what it does, rather than by what it is. Although not drawn to the DNA arts, the instant situation is clearly amendable to the type of analysis set forth in *Ex parte* Maize (27 USPQ2d 1662 at 1665) where it was found that:

> "Appellants have not chosen to claim the DNA by what it is but, rather, by what it does, i.e. encoding either a protein exhibiting certain characteristics or a biologically functional equivalent thereof. Appellants' claims might be analogized to a single means claim of the type disparaged by the Court of Customs and Patent Appeals in *In re Hyatt*, 708F.2d712, 218 USPQ 195 (Fed. Cir. 1983). The problem with the phrase "biologically functional equivalent thereof" is that it covers any conceivable means which achieves the stated biological result while the specification discloses, at most, only a specific DNA segment known to the inventor. Clearly the disclosure is not commensurate in scope with the claims."

Although the language of the instant claims does not recite the phrase "biologically functional equivalent thereof" and although the claims are limited to a polypeptide, implicit and explicit in the teachings of the specification is that ECPKA, the "surprisingly and unexpectedly discovered" PKA is a biologically functional equivalent of intracellular PKA. Applying the logic of *Ex parte* Maize to the instant fact pattern, it is clear that ECPKA is not claimed by what it is, but rather is claimed by what it does, that is it phosphorylates a PKA substrate, it is inhibited by

PKI, monoclonal antibodies that bind to intracellular PKA bind to ECPKA and it is a Type I PKA. Since the assay is performed on a protein that is taught by what it does, rather than by what it is one would not know how to make or use the claimed invention.

Further, Weber et al, (Cold Spring Harbor Conferences on Cell Proliferation, 1991, 8:125-140) specifically teach that it was well known in the art that the R and C subunits of cAMP-dependent protein kinases contain conserved structures allowing C and R subunits, even from unrelated species, to form heterologous holoenzyme hybrids (p. 125). The authors used an immunotitration assay using type-specific antibodies which exhibited varying affinities to corresponding heterologous mammalian R proteins and in spite of differences in cross-reactivity, the heterologous R proteins could be quantitated by immunotitration as well (p. 128, para 2). Additional previously unknown R variants were found (p. 130). In the absence of a way to distinguish between the claimed but undefined ECPKA and intracellular PKA, ectoPKA and variants thereof, one could not make or use the claimed invention. The specification does not teach how to make the invention because the specification does not provide any distinguishing characteristics of ECPKA which would enable one of ordinary skill to predictably distinguish between ECPKA, for example in a solid tumor sample, and other forms of PKAs as required by the claims. Further, in the absence of the ability to distinguish between ECPKA and other PKAs one would not know how to use the claimed invention. In view of the above, one of skill in the art would be forced into undue experimentation in order to practice the invention as claimed.

Further, although Applicant suggests and claims in claims 3 and 6 (given that controls are assayed not for protein concentration, but for enzyme activity) that

the increased activity of ECPKA in serum is due to increased expression of ECPKA from cancer cells, compared to normal cells, it is not possible to predict from the information in the specification whether or not the increased activity is in fact due to increased expression of ECPKA in cancer cells as compared to normal cells. (It is here noted that the exemplification of *in vitro* cell culture studies, with cancer cells altered with constructs for the constitutive expression of known PKA subunits. is not commensurate in scope with the claimed invention and the information gained therefrom does not provide a nexus between the recombinant *in vitro* and the *in vivo* environments.)

In particular, it is well known in the art that deregulation of protein kinases, that is increases in their activity, is associated with cancer phenotypes. However, those increases in activity are not limited to increases associated with overexpression of the enzyme subunits. A wide variety of alterations in the enzymes, their cofactors and effectors lead to unregulated activity and are known in the art. For example, SRC, a protein kinase, is mutated in a subset of advanced human colon cancers. This mutation eliminates a phosphorylation site that regulates enzyme activity, is activating, transforming, tumorigenic and promotes metastasis and results in high enzyme activity in colon cancer patients compared with normal control (see Irby et al, Nature Genetics, 1999, 21:187-190, abstract). Further, CDK4, a protein kinase, is mutated in a subset of melanoma patients. CDK4 binding with protein cyclin D promotes the passage of cells through the G1 checkpoint. This activity is regulated by protein p16. p16 controls cell growth by inhibiting the activity of the CDK4-cyclin D complex and stopping cells at the G1 checkpoint. The CDK4 mutation disrupts the cell growth-inhibiting effects of p16 by preventing p16 from binding to CDK4 at the G1 checkpoint. Further, in a

subset of melanoma patients it was found that p16 is mutated, preventing the encoded protein from exerting its regulatory effects on the CDK4 complex (see CDK4 identified as a Familial Melanoma Gene downloaded from http://www.skincancer.org/melanoma/cdk4.php which reports the findings of Wolfel et al, Science, 1995, 269:1281-1284) and Zuo et al, (Nature Genetics, 1996, 12:97-99, abstract). In addition, Stott et al (BioTeach, Oncogenes: The (Autosomal Dominant Evil) downloaded from http://www.bioteach.ubc.ca/Cell Biology/Oncogenes/) specifically teaches the range of mutations that can occur that lead to the transformation of proto-oncogenes to oncogenes. In particular, the reference teaches that Abl is a tyrosine kinase that requires cytokine stimulation to be activated. In its monomeric form Abl is inactive. When cytokines are present, two monomers form a complex and auto-phosphorylate resulting in activation. Conversely, the brc gene contains a dimerization motif, but no kinase activity. When a translocation occurs between the abl gene and the brc gene, the fusion product contains the dimerization domain of bcr and the kinase domain of Abl. Consequently, the fusion protein dimerizes in the absence of cytokine, resulting in a constitutively active tyrosine kinase and uncontrolled cell division. Thus, it is clear that cellular events other than overexpression are known to be responsible for increases of protein kinase activity. Further, it is also reasonable to hypothesize that the increased enzyme activity found in the serum of cancer patients could be caused as well by cancer-dependent decreases in the affinity of the R and C subunits, increased affinity for substrate, cancer-dependent increases in extracellular camp. Although Applicant hypothesizes that exemplified increased activity of ECPKA in serum of carcinoma cancer patients is due to increased expression of one or both of the subunits of ECPKA, given the above, it is just as

reasonable to hypothesize that the increased activity demonstrated in the serum of cancer patients is due to alteration of sites associated with allosteric effectors, reduced availability of inhibitor, reduced affinity for inhibitor, mutation of the catalytic subunit that leads to constitutive activation, altered affinity for regulatory subunit, altered affinity for substrate. Given the above, in the absence of objective evidence demonstrating that ECPKA is overexpressed in relevant biological samples, it could not be predicted that ECPKA is overexpressed or that the claimed method of diagnosing cancer comprising assaying for an elevated level of ECPKA would function as claimed with a reasonable expectation of success. Since the specification provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention would function as claimed with a reasonable expectation of success it appears that undue experimentation would be required to practice the claimed invention.

Again, as drawn specifically to claims 3 and 6, for the reasons set forth above, since it cannot be predicted that the elevated enzyme activity is associated with elevated protein levels of ECPKA, it is not clear how one could use the level of ECPKA activity in blood serum as a measure to determine that the level of ECPKA protein is elevated.

Finally, as drawn to claim 9, given that the specification in particular teaches that the ECPKA is present in serum in the free C subunit form, it is unclear, even if the C subunit form is indeed overexpressed it is unclear how the R subunit could be quantitated since it does not appear that the R subunit is in fact in the serum.

(B) As drawn specifically to the assay sample, the specification teaches that while any sample from the patient theoretically can be used in the assay, desirably

the sample is a fluid sample, preferably blood, in particular blood serum or urine (p. 9, lines 13-15). One cannot extrapolate the teaching of the specification to the enablement of the claims because Weber et al, *Supra* specifically teach that in renal tumors, only the regular RI(49) was found that there were no visible amounts of variants (p. 131, para 3). Thus it appears that, it would not be expected that the assay of a renal tumor sample would present with a novel PKA or an overexpressed novel R. Weber et al further teach that when lymphocytes from normal patients were compared to lymphocytes from patients with CLL it was found that total cAMP-dependent protein kinase activity was drastically reduced and that total R subunits were reduced to the same degree. Comparison of R-protein patterns revealed fundamental differences between CLL and normal lymphocytes wherein it was revealed that the regular RI(49) component represented a principle cAMP-binding protein in the CLL patients but variant forms of RI could not be detected (p. 133, last paragraph). Thus it appears that, it would not be expected that the assay of a CLL tumor sample would present with a novel PKA or an overexpressed novel R. Again as set forth above, since Type I PKAs are quantitated by assay of the R subunits, it cannot be predicted based only on the information set forth in the specification as originally filed and the prior art of record, that the method will function as claimed with the broadly claimed sample with a reasonable expectation of success.

Finally, there is no guidance on how, in a solid tumor sample, one would distinguish between internal PKA, ectoPKA and ECPKA given that no distinguishing characteristics of ECPKA have been supplied by the specification.

(C) As drawn to the broadly claimed cancer assay of claims 1-3, 5-9, the specification teaches that the present invention seeks to provide a diagnostic assay

for cancer (p. 3, lines 25-30). The specification exemplifies elevated phosphorylation activity in the serum of 348 carcinoma patients when compared to normal controls (p.37).

One cannot extrapolate the teaching of the specification to the scope of the claims because it is well known in the art that cancers comprise a broad group of malignant neoplasms divided into two categories, that is carcinomas and sarcomas. Carcinomas are epithelial cancers which originate in epithelial tissues while sarcomas develop from connective tissues and those structures that had their origin in mesodermal tissues (Taber's Cyclopedic Medical Dictionary, F.A. Davis and CO., Philadelphia, 1985, p. 274) wherein the cells are specialized to give rise to the connective tissues, that is bone, cartilage, muscle, the urogenital system and the vascular system (Alberts et al, Molecular Biology of the Cell, Garland Publishing, Inc, NY, 1983, pgs 821-822). Although the specification clearly demonstrates that a number of different carcinomas present with increased phosphorylation activity in plasma, Osband and Ross (Immunology Today, 1990, 11:193-195) teach that the biochemistry, antigenicity and metastatic potency of neoplastic cells show considerable variation and that there is an obvious heterogeneity of tumors not only between patients but even between metastatic sites within a single patient (p. 194, para 2). Clearly there is heterogeneity between the expression of markers within a single cancer type and even between primary and metastatic cells in the same patient. Thus, the finding that the same marker is found across a broad range of epithelial cancers is surprising and unexpected. Given the above and given the art recognized physical differences between epithelial cells and cells of connective and mesodermal origin, it can not be predicted, nor would it be expected that the same marker, that is the increased phosphorylation activity in serum, would also be

found in cancer types that were not of the same lineage as the epithelial cell types. The specification provides insufficient guidance with regard to this issue and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention would function as claimed, with any cancer cell type other than carcinoma, with a reasonable expectation of success.

(D) As drawn to the specifically claimed cancer types in claim 4, one cannot extrapolate the teaching of the specification to the enablement of the claims because although the specification teaches that the specific type of cancer detected by the presence of ECPKA can be subsequently or simultaneously determined by methods well-known in the art and teaches well known genetic or protein markers which are predictive of a particular cancer, the claims as currently constituted do not recite method steps drawn to the identification of a particular cancer. It is noted that although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. In re Van Guens , 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993). Thus, even if it were to be found that the novel ECPKA is overexpressed in serum of cancer patients and that the increased phosphorylation activity exemplified in the specification is associated with increased expression of novel PKA subunits, given that the specification clearly exemplifies the increased phosphorylation in serum over a broad range of cancer types one could not distinguish between those cancer types and practice the invention as currently constituted with a reasonable expectation of success.

The specification provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the claimed method would function as

currently claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

5. Claims 1-9 are rejected under 35 USC 112, first paragraph, as lacking an adequate written description in the specification.

Claims 1-9 are drawn to a method of diagnosing cancer by assaying for the presence of ECPKA protein. Although drawn to DNA arts, the findings in University of California v. Eli Lilly and Co., 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997) and Enzo Biochem, Inc. V. Gen-Probe Inc. are relevant to the instant claims. The Federal Circuit addressed the application of the written description requirement to DNA-related inventions in University of California v. Eli Lilly and Co., 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997). The court stated that naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material.

The Federal Circuit has recently clarified that a DNA molecule can be adequately described without disclosing its complete structure. See Enzo Biochem, Inc. V. Gen-Probe Inc., 296 F.3d 1316, 63 USPQ2d 1609 (Fed. Cir. 2002). The Enzo court adopted the standard that the written description requirement can be met by show[ing] that an invention is complete by disclosure of sufficiently detailed, relevant identifying characteristics ....i.e., complete or partial structure, other physical and/or chemical properties, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics. Id. At 1324, 63 USPQ2d at 1613 (emphasis omitted, bracketed material in original).

The inventions at issue in Lilly and Enzo were DNA constructs per se, the holdings of those cases are also applicable to claims such as those at issue here. A disclosure that does not adequately describe a product itself logically cannot adequately describe a method of using that product.

Thus, the instant specification may provide an adequate written description of the ECPKA, per Lilly by providing information as to what ECPKA consists of. Alternatively, per Enzo, the specification can show that the claimed invention is complete by disclosure of sufficiently detailed, relevant identifying characteristics, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics.

In this case, the specification does not describe the ECPKA required to practice the method of claim 1 in a manner that satisfies either the Lilly or Enzo standards. The specification does not provide the complete structure of any ECPKA, nor does the specification provide any relevant identifying characteristics of the novel ECPPKA. Although the specification discloses a single undefined ECPKA, this does not provide a description of ECPKA that would satisfy the standard set out in Enzo.

The specification also fails to describe the ECPKA by the test set out in Lilly. The specification only names a type of material generally known to exist, in the absence of knowledge as to what that material consists of. Thus, the specification does not provide an adequate written description of the ECPKA that is required to practice the claimed invention. Since the specification fails to adequately describe the product which is to be assayed, it also fails to adequately describe the claimed method.

6. Claims 1-9 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The claims are indefinite in the recitation of ECPKA as the sole means of identifying the protein product to be assayed. The use of laboratory designations only to identify a particular protein renders the claims indefinite because different laboratories may use the same laboratory designations to define completely distinct proteins. Amendment of the claims to include the limitations uniquely identifying the claimed invention will obviate this ground of rejection.

Objections

7. The disclosure is objected to because it contains embedded hyperlinks and/or other form of browser-executable code, in particular pages 13-14. Applicant is required to delete the embedded hyperlink and/or other form of browser-executable code. See MPEP § 608.01.

8. No claims allowed.

9. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Susan Ungar, PhD whose telephone number is (571) 272-0837. The examiner can normally be reached on Monday through Friday from 7:30am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Siew, can be reached at 571-272-0787. The fax phone number for this Art Unit is (571) 273-8300.

Susan Ungar
Primary Patent Examiner
April 14, 2005